Contemporary Management of Subdural Hematoma

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KEYWORDS

• Subdural hematoma • Medical management • Neurosurgery • Traumatic brain injury
• Guidelines • Middle meningeal artery embolization

KEY POINTS

- Acute and chronic subdural hematomas (cSDHs) represent distinct entities with different risk factors, etiologies, and treatment approaches.
- Both acute subdural hematoma (aSDH) and cSDH arise from trauma, but the mechanisms differ. While aSDH often results from tearing of bridging veins or injured cortical arteries, cSDH may evolve from minor trauma in the context of shearing of the dural border cell layer. Understanding the pathophysiologies that contribute to the formation and recurrence of hematomas provides insights into novel therapeutic paradigms.
- Surgical evacuation is the mainstay of treatment for aSDH, particularly in cases with significant clot thickness and/or midline shift. However, initial nonoperative management may be appropriate for selecting patients.
- The management of cSDH is evolving, with emerging evidence for some medical interventions such as statins and tranexamic acid. Middle meningeal artery embolization has revolutionized care for cSDH. Surgical drainage with the use of surgical adjuncts like drains and body temperature irrigation remain important considerations in patient management.

INTRODUCTION

Definition and Classification

A collection of blood in the space between the dura and arachnoid mater is called a subdural hematoma (SDH). SDH typically manifests as a crescent shape in noninvasive imaging modalities, traversing the adherent suture–dural junctions of the calvarium while being confined within the boundaries of the falx cerebri and the tentorium cerebelli (**Fig. 1**). SDH can manifest as either acute (aSDH) or chronic (cSDH), each representing distinct entities on a disease spectrum with varying risk factors, etiologies, and treatment paradigms.

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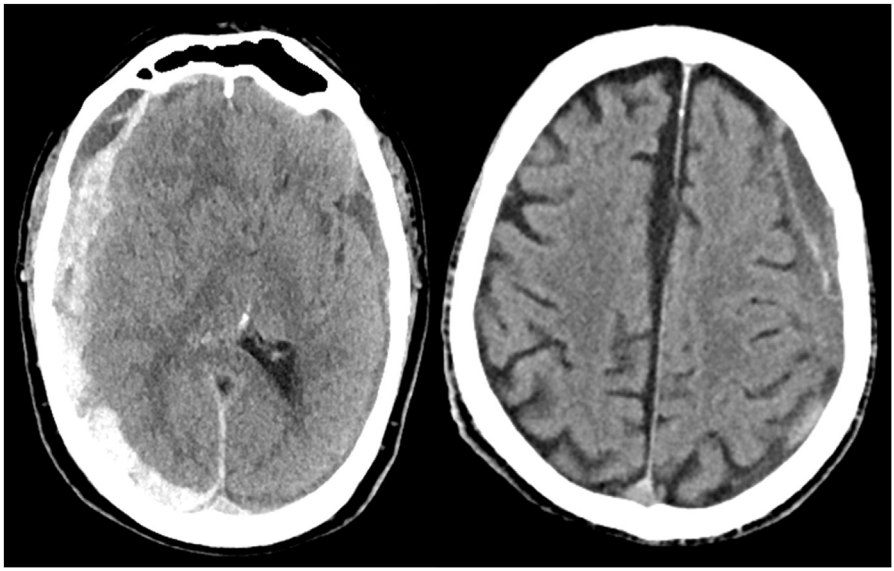

Fig. 1. *Left*: Right-sided subdural hematoma in an anticoagulated patient with mixed fluid density indicating acute and hyperacute blood products. *Right*: Chronic-appearing, isodense, left-sided subdural hematoma with a prominent hyperdense membrane coursing through the lesion.

Epidemiology

aSDH occurs in up to one-third of severe traumatic brain injury cases[1] whereas the incidence of cSDH has been reported to be between 1.7 and 79.4 per 100,000 persons per year.[2–4] The incidence of cSDH appears to be increasing over time, perhaps due to the more widespread use of noninvasive neuroimaging modalities, and is directly related to the rates of traumatic brain injury as well as increasing age.[5] Risk factors for aSDH include high-energy trauma, male gender, and age less than 50 years whereas risk factors for cSDH include advanced age (>60 years),[5] cerebral atrophy,[6] and blood thinning agents.[7]

Pathophysiology

Acute subdural hematoma. The most common etiology of aSDH is trauma, and the extent of superimposed traumatic brain injury contributes significantly to the clinical presentation and prognosis of the patient. The classic model for aSDH formation is tearing of bridging veins that are traversing the cortical surface into the dural sinuses. However, a significant number of aSDH are due to injured cortical arteries that bleed into the subdural space.[8] Given the considerably higher arterial pressure relative to intracranial pressure (ICP), it becomes readily apparent how a ruptured cortical artery could swiftly accumulate blood within the subdural space. Less obvious is how a torn vein, with its considerably lower cerebral venous pressure, could accumulate a substantial aSDH before being tamponaded by the clot itself and the resultant rising ICP.[9] It has been proposed that bridging veins are susceptible to shearing within the dural border cell layer of the dura mater.[10] This shearing leads to an unraveling of the delicate dural border cell layer. Paradoxically, the rising ICP would correspondingly elevate intracranial venous pressure,[11–13] thus enlarging the hematoma.[9] Therefore, despite the differences in venous and arterial pressure, aSDH from either a bleeding cortical artery or bridging vein may have a similar result.

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Chronic subdural hematoma. In general, cSDH is the result of trauma. While some cases of cSDH evolve from liquefaction of a known aSDH, in many cases of newly diagnosed cSDH, the preceding trauma is often so minor that patients may not recall an inciting event. Because cSDH is exponentially more prevalent in patients with advanced age, a possible link between age and cSDH is via cerebral atrophy, which may result in tension on the bridging veins, making them more susceptible to injury from relatively minor trauma.

The subdural space is bounded by dural border cells which, when sheared from even minor trauma, leak blood into the subdural space due to their thin bridging/traversing veins.[10,14,15] This expands the potential space and results in release of cytokines from the dural border cells, attracting fibroblasts, which form thick outer and thin inner membranes of the hematoma cavity. Due to a reactive inflammatory cascade and release of angiogenic factors, the outer membrane forms abundant, fragile blood vessels with highly permeable capillaries, leaking blood into the subdural space.[16] Blood in the subdural space clots and then eventually undergoes fibrinolysis,[17,18] which stimulates a local inflammatory response,[19–23] and results in recurrent hemorrhage into the subdural space from the leaky capillaries of the outer membrane.[24,25]

In a patient who presents with nontraumatic, bilateral subdural fluid collections that are chronic-appearing or of mixed density, evaluation for an occult cerebrospinal fluid leak may be necessary if the patient endorses typical symptoms, such as a clear postural (orthostatic) headache.

MANAGEMENT

Acute Subdural Hematoma

According to guidelines published from a panel of neurosurgical experts,[26] surgical evacuation is indicated for

1. Patients with an acute subdural hematoma with clot thickness greater than 10 mm or with midline shift greater than 5 mm on computed tomography regardless of the patient's Glasgow Coma Scale (GCS) score.
2. Patients with a GCS score less than 9 with an aSDH less than 10 mm thick and midline shift less than 5 mm if the GCS score decreases by 2 or more points between the time of injury and hospital admission and/or if the patient presents with asymmetric or fixed/dilated pupils and/or if the ICP exceeds 20 mm Hg.

When surgery is indicated for aSDH, it should occur without delay.

Not all cases of aSDH require emergent neurosurgical intervention, however. In a real world analysis of 869 patients presenting with aSDH, 74.3% were initially managed nonoperatively with only 6.5% of those patients requiring delayed surgery.[27] Patients appropriate for initial nonoperative management tended to have small (maximal thickness <10 mm) SDH, were asymptomatic, or were very elderly with minimal symptoms. Thicker SDH, greater midline shift, convexity location, history of alcohol abuse, and history of falls were associated with need for delayed surgery.

aSDH patients selected for nonoperative management are generally admitted to the intensive care unit for close neurologic observation.[27] ICP monitoring is indicated for those patients whose GCS score is 8 or less who have an abnormal scan (including an aSDH that does not meet strict operative criteria).[26] Prophylactic anti-epileptic therapy is indicated for a 7 day duration for patients with aSDH[28,29] using either levetiracetam or fosphenytoin.[30] In patients who are immobile, prophylaxis for venous thromboembolism can be initiated with either low-molecular-weight heparin or unfractionated heparin 24 hours after stable head computed tomography.[31,32]

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For patients with aSDH in whom emergent surgery is warranted, surgery may proceed with either a standard reverse question mark, retroauricular, or T-shaped Kempe incision based on surgeon's preference, history of prior scalp incisions, and need for future extracranial-to-intracranial bypass surgery.[33,34] Because aSDH often coincides with sequalae of severe traumatic brain injury, including parenchymal injury and diffuse cerebral edema, it is essential to plan for a large, unilateral frontotemporoparietal bone flap in accordance with the Brain Trauma Foundation TBI Guidelines.[35] When brain swelling after hematoma evacuation prevents safe replacement of the bone flap, a craniectomy is performed, necessitating cranioplasty for skull reconstruction weeks to months after the initial surgery. Because decompressive hemicraniectomy has been shown to reduce mortality in cases of refractory posttraumatic intracranial hypertension,[36] there has been some controversy[37] as to whether a craniectomy should be pre-emptively performed when evacuating aSDH in anticipation of delayed cerebral edema and elevated intracranial hypertension given the risks associated with cranioplasty.[38] In a recent multicenter, randomized, controlled trial comparing outcomes of craniotomy versus craniectomy in adult patients with traumatic aSDH,[39] the investigators found no difference in disability or quality of life outcomes between the groups but there was a higher rate of additional surgery (namely craniectomies) within 2 weeks of the initial surgery in the craniotomy group as compared to the craniectomy group (14.6% vs 6.9%), and a 3 fold increase in wound complications in the craniectomy group as compared to the craniotomy group (12.2% vs 3.9%).

Postoperatively, anti-seizure prophylaxis is generally continued for a total of 7 days. That said, seizures or epileptiform changes on electroencephalogram are observed during the acute postoperative period in approximately 25% of patients[40] and, in such cases, antiepileptic management is typically escalated and continued for a minimum of 3 months. Initiation of venous thromboembolism (VTE) prophylaxis is more precarious as compared to patients with aSDH managed nonoperatively. A large retrospective cohort study investigated the timing of VTE prophylaxis in adult patients after urgent neurosurgical intervention for TBI in relation to thromboembolic and intracranial complications.[41] Early initiation (during the first 3 postoperative days) of VTE prophylaxis was associated with reduced risk of thromboembolism but also increased risk of additional neurosurgical intervention, highlighting the delicate balance between intracranial bleeding and peripheral clotting in these delicate patients.

Chronic Subdural Hematoma

Management of patients with cSDH is much more nuanced due to the fact that many patients are elderly with multiple comorbidities, are often asymptomatic or minimally symptomatic, and because of additional interventions available for cSDH, particularly middle meningeal artery embolization (MMAe), which has revolutionized care for patients with cSDH. As cSDH is thought to derive from fragile, inflamed vascularized neomembranes supplied by the MMA,[42,43] non-operative interventions have been employed to target the inflammatory or angiogenic etiologies of cSDH.

Medical treatment of chronic subdural hematoma

The 3 most extensively studied medical interventions for cSDH are corticosteroids, atorvastatin, and tranexamic acid (TXA).[44] Steroids are thought to inhibit the formation of neomembranes and neovessels and systematic reviews suggested that steroids may prevent the need for surgical intervention and reduce the risk of recurrence after cSDH drainage.[45,46] In 2020, a multicenter, randomized trial aimed to determine the effects of dexamethasone treatment on outcomes in patients with symptomatic cSDH.[47] Of the 748 patients enrolled, 94.2% underwent surgical evacuation of the

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hematoma. Fewer patients in the group treated with steroids reported a favorable outcome (modified rankin scale [mRS] 0–3) as compared to the control group (83.9% vs 90.3%); however, the incidence of repeat surgery for hematoma recurrence was much lower in the dexamethasone group as compared to the placebo group (1.7% vs 7.1%). The incidence of serious adverse events was greater in the dexamethasone group as compared to the placebo group (16.0% vs 6.4%). An additional multicenter, randomized trial was published in 2023 that sought to determine whether dexamethasone as a stand-alone therapy had the same potential to achieve favorable outcomes as surgical evacuation in patients with symptomatic cSDH.[48] This trial was stopped early because of safety and outcome concerns in those patients treated with steroids. Specifically, the odds ratio for an mRS 0 to 3 at 3 months with steroids versus surgery was 0.55 (95% confidence interval, 0.34–0.90), complications occurred in nearly twice as many steroid-treated patients (59% vs 32%), and the need for additional surgery was 9 times greater in the steroid group (55% vs 6%). Thus, steroids are not recommended for use in cSDH.

Statins, whose canonical function is to reduce low-density lipoprotein cholesterol in patients with hyperlipidemia, are also thought to inhibit inflammation and immature angiogenesis in the neomembranes of cSDH.[49] The results of a multicenter, randomized, phase II clinical trial investigating the safety and efficacy of 20 mg atorvastatin daily for 8 weeks versus placebo in cSDH treated nonoperatively were reported in 2018.[50] Patients in the atorvastatin group saw greater reduction in hematoma volume, had significantly improved neurologic function, and underwent less surgery for enlarging hematoma and/or deteriorating neurologic condition, together suggesting a possible role for atorvastatin in the nonsurgical treatment of patients with cSDH. The phase III trial is currently underway (NCT03956368).

TXA inhibits fibrinolysis by blocking the lysine receptor on plasminogen and plasmin. There have been reports of TXA being safe and effective for the treatment of cSDH both as stand-alone therapy[51] and in combination with surgery.[52] As to the latter point, an observational study with propensity score matching of patients undergoing surgery for cSDH showed that the reoperation rate was markedly lower in patients treated with TXA (1.9% vs 6.1%) with no difference in adverse events. There is one active and one pending clinical trial investigating the role of TXA in cSDH hematoma: the TXA to prevent operation in chronic subdural hematoma (TORCH) trial[53] (NCT03582293) and the use of TXA in the treatment of symptomatic subdural hematoma (TRACE) trial (NCT05713630).

Middle meningeal artery embolization

Few interventions have revolutionized the management of any medical ailment the way that MMAe has impacted care of patients with cSDH.[54] While MMAe has gained widespread traction over the past 5 years, the first reported case of MMAe was performed in Japan in 2000.[55] The zeitgeist at the turn of the century was that cSDH originated from venous bleeding. Therefore, the rationale to embolize an artery—particularly the MMA, which is implicated in epidural rather than subdural hematomas—in the treatment of cSDH was, by all accounts, illogical. Yet the pioneers of MMAe were able to avoid being trapped by dogma and create a paradigm shift by combining 3 key observations. First, diffuse dilation of the ipsilateral MMA with scattered abnormal vascular networks was seen in patients with cSDH who underwent catheter angiography.[56] Second, cSDH accumulates contrast material administered systemically, suggesting a connection between the systemic vasculature and the hematoma.[55] Third, MMAe was an established procedure for preoperative embolization of meningiomas and, thus, interventionalists were familiar with the approach and safety had already been established.

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The first large case series of MMAe for cSDH was published in 2017, comparing 72 prospectively enrolled patients with cSDH who underwent MMAe either as stand-alone therapy or in conjunction with surgery against a historical control group of 469 patients who were either managed with close observation or surgery.[57] The authors reported spontaneous resolution of all asymptomatic patients who underwent stand-alone MMAe and a marked reduction in need for rescue surgery in patients receiving MMAe as compared to the historical control cohort (1.4% vs 18.8%). Since then, there has been an explosion of articles published about MMAe for cSDH.[54,58] A recent systematic review and meta-analysis analyzed outcomes in cSDH patients treated with MMAe to those without.[59] The authors identified 9 observational studies including 1523 patients of which 337 received MMAe. They reported a significant reduction with MMAe versus conventional management in terms of treatment failure (5.6% vs 22.2%) and need for surgical rescue (4.1% vs 16.1%), higher chance of complete hematoma resolution with MMAe versus conventional management (61.9% vs 26.4%), and no difference in complication rates, hospital length of stay, functional outcomes, or mortality. The results of 3 major trials (EMBOLISE, MAGIC-MT, and STEM) were recently presented at a major international meeting.[60] Based on the data presented, these studies are the first Level 1A evidence to show that MMAe, whether used alone or as a surgical adjuvant, prevents cSDH recurrence or progression and significantly reduce the need for repeat cSDH drainage and should therefore be routinely used in the care of patients with cSDH.

There has been growing interest recently as to the safety, feasibility, and efficacy of MMAe across multiple classes of embolic materials including liquids, particles, coils, and combinations thereof.[61] Polyvinyl alcohol (PVA) particles, flow-directed materials that result in mechanical obstruction of the artery, have been the most common embolic agent used in studies investigating MMAe for treating cSDH.[62] PVA particles have a few distinct advantages. First, as they are flow-directed, the microcatheter can sit in the proximal MMA or just distal to the foramen spinosum where the outer diameter of the microcatheter is less than the inner diameter of the artery to facilitate distal flow of the particles. Second, PVA particles are small enough to travel into the distal MMA branches yet large enough to avoid entering the dangerous extracranial–intracranial (EC-IC) anastomoses of the MMA,[63] namely collateralization with the internal carotid artery through its cavernous branch, ophthalmic anastomosis via the lacrimal artery, and facial arcade by way of the petrosal branch. Third, procedural pain is generally minimal with particle embolization of the MMA and can be performed in most patients without general anesthesia. However, PVA particles tend to aggregate proximal to the intended target, which can result in distal recanalization through neoangiogenesis and they are radiolucent and cannot be directly visualized. These particles may also reflux since the microcatheter is not wedged into the distal MMA.

Liquid embolic agents (Onyx, N-butyl-2cyanoacrylate/TruFill, Squid, PHIL), on the other hand, address many of the disadvantages of particles. Liquid agents tend to penetrate much deeper into the vessel than particles, they result in a more durable embolization, and they are opacified, allowing the operator to see exactly where the embolisate is at all times. However, liquid agents can be pushed through angiographically invisible EC-IC anastomoses, and can lead to stroke, blindness, and/or permanent facial nerve paralysis if the microcatheter is not wedged beyond these tiny collaterals. Furthermore, many of the liquid agents require vehicle delivery in dimethyl sulfoxide, which can be painful for patients, necessitating either general anesthesia or conscious sedation. An active area of interest and investigation is comparing the safety and efficacy of particle versus liquid agent embolization for patients with cSDH, although preliminary analysis seem to indicate that both agents are safe and effective with nearly identical outcomes.[64–68]

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Surgery

Traditionally, surgery for cSDH has included twist drill craniotomy, burr hole craniostomy, or craniotomy/mini-craniotomy.[69] The goal of each procedure is the same—to evacuate the hematoma—but patient-specific, hematoma-specific, and/or surgeon-specific factors may influence the selection of one technique over the others. Twist drill craniostomy is a bedside procedure performed under local anesthetic often reserved for patients who are otherwise not appropriate for general anesthesia and for patients with highly liquefied cSDH without many intervening membranes. Burr hole craniostomy is the most frequently used and most studied surgical intervention for patients with cSDH.[69] Usually 2 large (>2.5 cm) burr holes[70] are drilled over the cerebral convexity followed by a cruciate durotomy to access the subdural space. Copious irrigation is then used to clear the subdural space of hemorrhagic debris. Finally, a conventional craniotomy or mini craniotomy may seldom be needed for subdurals with extensive membranes or for mixed hematomas with a significant acute component. Although most neurosurgeons use the 2 burr hole craniostomy technique, multiple systematic reviews and meta-analyses have failed to consistently demonstrate clear superiority of any one technique[69,71,72] and, therefore, treatment can be tailored to the individual patient and clinical scenario.

There are 2 surgical adjuncts that have been shown to significantly reduce hematoma recurrence after cSDH surgery: drains and body temperature irrigation. In 2009, a randomized controlled trial of adult patients undergoing burr hole drainage of cSDH demonstrated clear superiority of subdural drain placement as compared to no drain.[73] The authors found that 9.3% of patients with a subdural drain had a recurrent hematoma requiring redrainage versus 24.0% of patients without a drain. Additionally, patients who had a subdural drain placed had a significant reduction in mortality at 6 months postoperatively (8.6% vs 18.1%). Because placement of a subdural drain may be impossible in cases of rapid brain re-expansion or predispose to complications, namely injury to the brain parenchyma, various groups have investigated the utility of alternatively placed drains in the subgaleal/subperiosteal space[74,75] and have found subdural and subgaleal/subperiosteal drains to be interchangeable[76] in terms of reducing hematoma reaccumulation and mortality. A recent multicenter, randomized trial investigating the relationship between subdural drainage time and 3 month SDH recurrence found a 27% recurrence rate with 6 hours of postoperative drainage as compared to 20% with 12 hours and 10% with 24 hours, thus establishing 24 hours as the optimal postoperative drainage time following cSDH evacuation.[77] Warm, body-temperature irrigation fluid in the evacuation of cSDH has also been shown to significantly reduce hematoma recurrence.[78] In a 2023 multicenter randomized clinical trial of adult patients who underwent cSDH evacuation, patients in the body temperature irrigation fluid group had a significant reduction in recurrent hematoma requiring reoperation as compared to those with room temperature irrigation (6% vs 14%). These studies strongly suggest that in patients undergoing cSDH surgery, leaving a drain postoperatively (either a subdural drain or a subgaleal/subperiosteal drain) and body temperature irrigation intraoperatively should be standard of care.

Decision-making in chronic subdural hematoma

Unlike aSDH, there are no expert guidelines for the management of cSDH but some helpful contemporary decision algorithms for cSDH management have been proposed.[79] Prior to the widespread use of MMAe, most cSDHs were considered for surgery if the hematoma thickness exceeded 1 cm and/or midline shift exceeded 5 mm regardless of patient presentation. In the era of MMAe, however, there is a stronger focus on symptoms to decide whether surgery is needed. MMAe has shown to be

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very safe and effective and can serve as the primary treatment of patients with asymptomatic or mildly symptomatic cSDH who can be monitored closely outside the hospital. Indeed, most cSDHs at our institution get MMAe unless the cSDH is found incidentally, the patient is completely asymptomatic, and the cSDH is small without any local mass effect. For patients who are symptomatic from their cSDH and/or have a cSDH that results in at least 5 mm of midline shift and/or if there is cortical flattening due to the size and mass effect of the cSDH, surgical evacuation is appropriate. In these cases, surgery should be performed with body temperature irrigation fluid, a subdural or subgaleal/subperiosteal drain should be placed, and, at centers with appropriate capabilities, adjuvant MMAe should be performed.

SUMMARY

aSDH and cSDH represent distinct entities on a disease spectrum with different epidemiologic patterns, risk factors, and treatment approaches. For aSDH, timely surgical intervention remains crucial, guided by established criteria such as clot thickness, midline shift, and neurologic status. However, nonoperative management may be an option for selecting patients, highlighting the importance of close monitoring and individualized decision-making. In contrast, the management of cSDH requires a more nuanced approach, considering the evolving therapeutic options. While surgical evacuation remains a cornerstone, recent advancements such as MMAe have transformed treatment paradigms, offering a less invasive yet highly effective alternative or adjunct to surgery. Additionally, medical interventions, namely statins and TXA may have a role in the future management of cSDH patients pending the results of ongoing clinical trials.

CLINICS CARE POINTS

- Management of acute subdural hematoma requires emergent evaluation by a neurosurgeon.
- Chronic SDH management has evolved over time, with middle meningeal artery embolization being more commonly used in these patients.
- Ultimate management for subdural hematoma requires review by a neurosurgon to determine if operative or non-operative management is appropriate.

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